

higher levels of intake than 1-4 ppm."⁴ They further state that crippling fluorosis "... results from the continuous exposure of an individual to 20-80 mg of fluoride ion daily over a period of 10-20 years. Such heavy exposure is associated with a level of at least 10 ppm in the drinking water supply." These fluoride levels do not exist in the US, and there have been no reported cases of crippling fluorosis in the United States.

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Mr Russell and Drs Jackson and Spath respond

TO THE EDITOR: We thank Dr Isman for his response to our article and will clarify our comments on the hazards of excess fluoride in drinking water.

First, we erred in our paraphrasing of the American Academy of Pediatrics Committee on Nutrition statement on fluoride supplementation. The committee reported: "The optimal systemic fluoride dose to prevent caries appears to be .05 to .07 mg per kg per day. The narrowness of the therapeutic range is emphasized by the fact that mild fluorosis has been seen with oral intakes greater than .1 mg per kg per day." Note that this is a dose per bodily weight, not per liter of water. Dr Isman has correctly quoted the ideal concentration in public water supplies as 0.7 to 1.0 ppm (mg/L).

Second, we evidently selected the wrong reference from a much longer staff report when we stated that skeletal fluorosis has been observed at 3 ppm in drinking water. A recent literature survey reveals that many authorities reported that skeletal fluorosis may occur at levels above 3 ppm or above 4 ppm, though bone changes can be detected at lower levels. For example, the World Health Organization (WHO) stated, "Skeletal fluorosis has been observed in persons when water contains more than 3-6 mg of fluoride per liter depending on intake from other sources."¹ It is well recognized that the total fluoride intake is affected by factors such as climatic conditions, amount of drinking water consumed, fluoride intake from sources other than drinking water, food habits, and malnutrition, which would influence the development of fluorosis.² A second WHO report noted that in tropical countries where dietary intake of fluoride from other sources is high, "Relatively marked osteofluorotic symptoms were connected with fluoride levels as low as 1-3 mg/litre drinking water."² The National Academy of Sciences stated that "Skeletal fluorosis has been observed with use of water containing more than 3 mg/liter,"³ and the Environmental Protection Agency reported that "Bone changes (increased bone density; calcification of sacrospinous and sacrotuberous ligaments) described in this study [a study of bone density in Texas and Oklahoma] were found when the drinking water contained 4-8 mg/L."⁴ A Finnish study of bones from cadavers reported histomorphometric bone changes at 1.5 ppm.⁵

WHO has stated that daily intake exceeding 8 mg (equivalent to 4 mg per liter in water at 2 liters a day consumption) is suggested to be harmful in adults. WHO recommends an upper limit of 1.5 mg per liter in drinking water. A recent study in Senegal, however, found that in a hot dry climate where water intake is high, the WHO limits did not protect adequately against crippling skeletal fluorosis or dental fluorosis, and that for such climates the limits should be set lower.⁶

Our concern with the Environmental Protection Agency's raising of the federal primary drinking water standard from a maximum of 2.4 to 4 mg per liter is that dental mottling could occur in children, especially if the secondary (voluntary) standard of 2 mg per liter is not adhered to. The National Research Council has stated that dental mottling, depending on temperature, "may occur to an objectionable degree with fluoride concentrations . . . of only 0.8-1.6 mg/liter [ppm]."³ California has therefore retained the earlier primary limits ranging from 1.4 mg per liter in hotter climates to 2.4 mg per liter in cooler climates to try to prevent objectionable and potentially psychologically damaging mottling and pitting of the teeth of children and adolescents.

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Pediatric Liver Cancers

TO THE EDITOR: The February 1988 article by Tong and Govindarajan prompted me to write.

During the years 1965 to 1970, I worked in a mission hospital in Kenya, Africa. Our 120-bed hospital treated very many cases of acute hepatitis with jaundice. Also, several times each year we admitted a small child with liver cancer. The livers were firm and multinodular. No biopsies were done.

Since it is now known that hepatitis B is common in Africa, it is possible that these malignant lesions were due to hepatitis B virus. A long-term study in Africa should add much to our knowledge of the epidemiology of these pediatric liver cancers.

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